average alignment of fields with fiber orientation or to some combination of these factors.

8:45

738-2 Postshock Epicardial Activation Patterns Predict Outcome of Attempted Defibrillation

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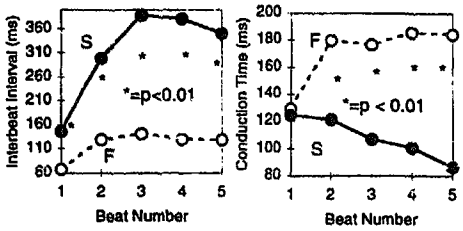
Recent work has shown that organized ectopic wavefronts arise from regions of myocardium near the apex following attempted near-threshold defibrillation (DF) with endocardial leads. We hypothesized that the epicardial activation patterns (EAP) for these postshock ectopic beats would differ for failed (F) and successful (S) shocks. In 6 dogs the heart was exposed and a 510 electrode shock with ≈4 mm interelectrode spacing was pulled over the ventricles and sutured to the AV groove. DF coils were placed in the RV apex and SVC. Unipolar epicardial electrograms [V(t)] were recorded using a 528-channel cardiac mapping system. Shocks were delivered after 10 sec of VF at shock strengths scanning the DF range in 100V steps. EAPs following the DF shocks were recorded and analyzed by animating the dV/dt. Interbeat intervals (time between successive postshock ectopic beats) and wavefront conduction time (time between the earliest and latest activations of an ectopic beat) were determined for the first 5 postshock beats. Sinus beats were not included in the analysis. Mean interbeat intervals were significantly longer for S than F for all 5 beats. Mean wavefront conduction time was significantly shorter for S than F for beats 2–5.



Conclusion: Postshock ectopic EAPs are similar for the first beat following F and S shocks, but then become increasingly dissimilar. Prolonged conduction time and shorter interbeat intervals, which create overlapping beats on the heart, may facilitate reentry and lead to VF.

9:00

738-3 Strong Electrical Stimulation Increases Intracellular Calcium in Perfused Rabbit Hearts

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Studies with isolated cardiac cells have indicated that strong shocks (S) produce membrane pores through which calcium (Ca) can enter cells. Increased intracellular Ca after defibrillation S, if it occurs in the heart, may be important for dysfunction and post-shock arrhythmias. We have mapped intracellular Ca-sensitive fluorescence after S in isolated perfused rabbit hearts. *Methods:* Hearts (n = 5) were intracellularly loaded with fluo-3 acetoxymethyl ester by perfusion at 5–10 μM for 20 min. A laser beam having a wavelength of 488 nm scanned 63 recording spots between and 1.1–4.6 mm from mesh S electrodes on the anterior ventricular epicardium. Fluorescence having wavelengths > 515 nm was recorded. The Ca transients increased with heart rate, decreased after verapamil administration and resembled transients reported by others. Control Ca transients were recorded during sinus beats before S. Truncated exponential S with capacitance of 140 μF, duration of 8 ms and leading edge of 100, 150 or 300 V were given. *Results:* The mean ± sd changes in diastolic Ca 5 sec after S at different distances from S electrodes are tabulated as multiples of heights of control Ca transients. Diastolic Ca increased significantly after S of 150 and 300 V. The Ca increase was greater at 1.1 mm away from S electrodes compared to 2.3–4.6 mm away.

Leading edge	1.1 mm	2.3 mm	3.4 mm	4.6 mm
100 V	1.1 ± 1.1	0.7 ± 0.8	0.3 ± 0.3	0 ± 0
150 V	3.1 ± 3.5*	1.9 ± 2*†	1.3 ± 1.3*†	1 ± 0.7*†
300 V	7.8 ± 4.9*	6.8 ± 3.5*	5.4 ± 4.4*†	4.5 ± 3.4*†

*p < 0.05 vs no change, †p < 0.05 vs change at 1.1 mm for same S

Conclusions: 1) Strong S can increase intracellular Ca in rabbit hearts. 2) The Ca increase is largest near S electrodes.

9:15

738-4 Intracardiac Defibrillation-Strength Shocks Produce Large Regions of Hyperpolarization and Depolarization

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Certain mathematical models predict that strong shocks (S) produce many small regions of hyperpolarization and depolarization interspersed in the myocardium. However, little experimental evidence exists to confirm these predictions. The study purpose was to examine regions of hyperpolarization and depolarization during an intracardiac S. *Methods:* Isolated rabbit hearts (n = 6) were perfused with Tyrode's solution and stained with the voltage-sensitive dye, di-4-ANEPPS. Hearts were paced (s1) at a cycle length of 250 ms at 2x diastolic threshold with a bipolar Ag-AgCl electrode on the lateral left ventricular epicardium. A 2 A square-wave shock (S2) with a 20 ms pulse width was delivered during the plateau (Phase 2) of the action potential (AP) through two intracardiac 1.0 cm titanium electrodes at the right ventricular (RV) apex and right atrium. An optical laser scanner recorded transmembrane potentials at 63 spots of a 1.6 cm × 1.6 cm grid projected over the anterior left and right ventricles. The locations of laser spots that were polarized by > 5% of the maximum AP amplitude during the S2 shock were determined. *Results:* There was an increased number of regions hyperpolarized than depolarized (2.0 ± 0.39 vs 1.0 ± 0.89, p = 0.012) during anodal RV shocks but not during cathodal RV shocks (1.33 ± 0.82 vs 2.17 ± 0.75, p = 0.093). Hyperpolarized regions encompassed a larger area than the depolarized regions (1.18 ± 0.77 cm^2 vs 0.30 ± 0.32 cm^2, p = 0.028). *Conclusion:* Defibrillation-strength S produce large regions of either polarization. Hyperpolarization occurs more frequently and occupies a larger area than depolarization. These results are contrary to models of transmembrane potential changes during a shock.

9:30

738-5 Regional Capture of Fibrillating Ventricular Myocardium With Periodic Anodal Stimulation: How Excitable is the Excitable Gap?

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Recent work suggests that regional capture (RC) of > 5 cm^2 of fibrillating right ventricular epicardium is produced by rapid, periodic, markedly suprathreshold electrical stimulation (15–25x diastolic pacing threshold). Our objectives were to determine if RC is possible with lower strength stimuli (STIM) and to characterize the excitability of the excitable gap during ventricular fibrillation (VF). Unipolar epicardial electrograms [V(t)] were recorded from 504 sites oriented in a rectangular grid (21 × 24, 1 mm spacing) surrounding a point stimulating electrode embedded near the center of a plaque attached to the antero-basal RV. Two bursts of 40 STIM were applied to the stimulating electrode (0.33 mm dia.) about 3 and 10 s after VF was electrically induced. Each STIM strength (1x, 2x, 5x, 10x, 15x, 20x, 30x diastolic threshold) was tested in random order in each of five pentobarbital anesthetized pigs (30–35 kg). STIM cycle length (CL) was constant for each animal (95% of mean VF CL over 60 activation intervals from 3 control episodes). Patterns of epicardial activation were reconstructed by animating dV/dt. *Results:* Sustained RC was observed in 37/140 VF episodes. The incidence of RC for each STIM strength [1x(0), 2x(4), 5x(8), 10x(7), 15x(7), 20x(5), 30x(6)] seemed to reach a plateau for STIM strength ≥ 5x. In all 5 animals, RC was observed (1) for STIM strength ≥ 5x diastolic pacing threshold and (2) more frequently during second STIM burst compared to the first burst (30 vs. 7). RC during VF was produced by relatively weak periodic anodal STIM. Onset of RC appears to depend on temporal confluence of STIM strength, ratio of STIM CL to VF CL (0.99 ± 0.08) and spatiotemporal complexity of myocardial activation patterns during VF. RC is possible with STIM only 5x > threshold. These data suggest that the excitable gap in VF may be partially but not fully excitable.

9:45

738-6 Reduced Arrhythmogenicity of Biphasic VS. Monophasic T Wave Shocks: Implications for Defibrillation Efficacy

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Background and Methods. Defibrillation energy is known to be lower for biphasic (Bi) than for monophasic (Mo) shocks. The underlying mechanism, however, remains unclear. The upper limit of vulnerability (ULV) hypothesis implies lack of initiation of ventricular fibrillation (VF) and suggests a similar